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A CASE OF SPINAL PARALYSIS IN AN ADULT, RESEMBLING THE SO-CALLED INFANTILE PARALYSIS.[1]

BY D. F. LINCOLN, M. D.

In the present state of science there can be no doubt of the propriety of establishing a class of spinal paralyses, dependent on a lesion of some of the large motor cells in the anterior cornua of the spinal cord. Most of the cases of this class occur between the ages of six months and two years; hence the trivial designation, infantile paralysis. A few, however, certainly occur in adult life. Duchenne was the first to recognize this latter fact, and the four cases published in the last edition of his "Électrisation localisée" were the first that were distinctly classed with "infantile" paralysis.

The name given by Duchenne, though expressive, is unfortunately very clumsy: *Paralysie spinale antérieure aiguë de l'adulte par atrophie des cellules antérieures de la moelle.* Frey[2] has proposed the shorter name "*Poliomyelitis anterior*," qualified by the addition of *acutissima* or *subacuta*, *adultorum* or *infantum*, as the case may be.

Before describing the present case, it will be well to give a brief summary of the essential symptoms of the disease, as we meet with it in young children. These symptoms are: A very rapid, almost sudden development of the palsy in its full extension; a subsequent wasting of the stricken muscles, usually with fatty degeneration; a loss of the power to react under the stimulus of induced electricity, with peculiar reactions under that of galvanic currents. There is a tendency to partial improvement; but if the improvement is only partial, deformities are apt to follow. There is an entire absence of symptoms referred to the bladder or rectum, to the muscles of respiration, or to the facial muscles; there is no tendency to atrophic changes of the skin and nails, or to the formation of bed-sores; and symptoms referable to irritation or paralysis of sensory nerves are very unusual. Thus infantile paralysis, in at least the great number of cases, is a pure motor paralysis, exclusively of spinal origin. We will now proceed to the case in hand.

G. E. B., a hardware merchant, aged forty-nine, a tall, stout man,

[1] Read before the Boston Society for Medical Observation.

[2] Berliner klinische Wochenschrift, 44, 1874. See also Medical Times and Gazette, January 23 and 30, 1875.

at the time of the first visit presented the appearance of tolerably good health. He had never had rheumatism or syphilis or any affection of the heart; was never subject to headaches or vertigo; never had an attack in any way resembling the present one. His sight and hearing were unimpaired. His mode of life was very regular and moderate, without exposure to any special injurious influences. His father and one of his sisters has fallen victims to what appears to have been cerebral hæmorrhage, at the ages respectively of fifty-four and fifty-three. When attacked, he had just returned from a vacation-trip of three weeks in the British Provinces; he had not been exposed to cold or rain or any other known source of disease, and was feeling unusually well.

On the 6th of August, 1873, he went to his business as usual. During the forenoon he noticed a feeling in his legs as if they had fallen asleep. This feeling came on again and again; and with it he began to be a little weak in the legs. Recollecting that his father and sister had died of paralysis, he was expecting an attack all the morning, although nothing beyond these premonitions occurred until about two o'clock. At that hour he was on his way to the station, and hailed a horse-car, but on trying to step upon the platform found that he could not do it. He was helped in, rode to the station, and thence by train to his home in the suburbs; was helped into a carriage, and driven to his house, where he was still able (with assistance) to walk up-stairs to his bedroom, but when seated was unable to rise from the chair. He was got to bed, and stayed there till I saw him, two days later; the weakness increased somewhat after this, but not much.

When seen by me on the 8th, two days later, his condition was but little altered. It was evident, in the first place, that the attack was not of cerebral origin. There was not the slightest evidence of any mental disturbance, nor has there been any up to the present day. He had not so much as felt giddy. His spirits were cheerful, and his mind active. The muscles of the face and eyeballs were under perfect control; the pupils were normal in size, and contracted well. Speech was natural. Vision and hearing were without defect.

The bladder and rectum performed their functions normally. The senses of touch, pain, and temperature were normal in the hands, and nearly so in the feet. Reflex contractions could hardly be obtained from the soles. There was a complete absence of abnormal sensations; the tingling he had felt on the day of the attack had passed off, and did not reappear; there was no sensation of numbness or constriction, no muscular twitching or trembling; he felt no pain anywhere, and the spine when percussed was not tender. The pulse was 80, soft and regular; the heart-sounds were normal, temperature in axilla was 98°. The urine was tested a few days later and found to be free from albumen.

The muscles of the neck and limbs, except below the knees, were generally in a condition of semi-paralysis. He lay on his back, almost helpless; could not raise his head from the pillow without some help, could not raise his knees from the bed by flexing the thighs, and the grasp of his hand was very feeble indeed. There was no absolute paralysis of any muscle. Below the knees he seemed to have his full strength. The weakness was much more marked on the left side than on the right.

His condition remained as thus described during the six months of his confinement to the house, except that the paresis of the muscles gradually disappeared. A slight sense of muscular fatigue in the shoulders should be mentioned; this soon left him. Loss of appetite was relieved by nux vomica and cinchona, and subsequently by tincture of iron with strychnia, and Horsford's acid phosphates of lime and magnesia.

On the fifth day of the attack, treatment by the induced electric current was begun, when it was found that the muscles, some of them at least, had already lost a part of their susceptibility to this stimulus. The loss went on increasing; within a week or ten days from the first attack it was perceptible in all the paralyzed muscles, and in some of them it afterwards appeared to become total for a short time. By the twenty-first day the loss had become so great that the galvanic current[1] was substituted for the faradic in the hope of obtaining a more satisfactory reaction. It was found that the galvanic reaction was also diminished, though to a less extent than the faradic; for example, the rectus femoris of the left side required forty cells to produce a feeble contraction, while twenty cells acted strongly upon the corresponding muscles of the son of my patient, a vigorous young man. The galvanic treatment then adopted, and continued until his recovery, consisted in the application of descending current to the spine, and of labile and interrupted currents to the muscles, three times a week; the faradic current was also continued for a few weeks. I subsequently found much advantage in using a hot-air bath long enough to bring my patient into a profuse sweat, directly before making these applications. In these measures, and in the various simple gymnastic exercises which he was able to perform with assistance, I was greatly assisted by his son, a student of medicine.

The reaction to both orders of electric currents seemed extinct in the recti femoris muscles on the twenty-fifth day, but this was partially due to a degree of weakness in the fluids of the batteries; improvement, however, seemed to commence from that period, and to go on steadily.

[1] From one of Hirschmann's portable zinc-carbon batteries (worked with bisulphate of mercury), of which the action nearly corresponded with that of an equal number of Daniell's cells.

The trapezii, glutæi, and sartorii, were at least equally reduced; the sterno-mastoids reacted rather better, but it was impossible to find any superficial muscle in the neck, shoulders, arms or thighs, that reacted quite well. There was, however, no difficulty in swallowing or speaking, and the act of respiration was performed easily. In each muscle the power to respond to the galvanic shock seemed to improve very rapidly, however low it had fallen at the time of commencing the special treatment. As regards the reactions of the nerves, my observations were defective: it is noted, however, that the full strength of the faradic battery produced but a slight muscular contraction (flexores digitorum) when the negative pole was placed on the left brachial plexus, above the clavicle, and none at all when placed on the right, the positive being put on the back of the neck. This on the sixty-seventh day, when he had lost a good deal in weight, and the deep parts of the neck were very accessible. The median and ulnar nerves reacted perfectly in the fore-arm at a somewhat earlier period. The "exaggerated reaction" was never observed in nerves or muscles.

The paralyzed muscles wasted, as is usual; in the course of six weeks this became very marked, and soon afterwards (before the fifty-second day) it is recorded that the hands began to tremble when held out, the balls of the thumbs being first affected. This tremor did not increase much, and at present, after the lapse of fourteen months, is not troublesome. His handwriting is not altered, though he cannot write as fast as formerly. The right hand now grasps the dynamometer with a force registered by 70, which is more than one half of the normal register for a person of his habits. On the fiftieth day it marked only 25, on the fortieth 20, and much less on previous occasions. His general improvement was very gradual, and it was six months before he was able to ride out. He resumed his business by degrees, and is now attending to it pretty much as before the attack, retaining his old habit of standing at his desk instead of sitting. He has lost twenty-five pounds in weight, chiefly of fatty tissue. The muscles are somewhat flabby, but not remarkably so for a man of his pursuits. The general health remains quite good, and the diminution in the weight of his body is regarded by him with satisfaction rather than the reverse.

In making the diagnosis of this case we can at once exclude the following affections: —

1. Progressive muscular atrophy. Not to speak of the totally different mode of invasion and of progression, the electric reaction by itself would be conclusive evidence against this diagnosis.

2. Lesion of the encephalon. There was no evidence whatever to indicate such lesion.

3. Acute myelitis. Under this term we commonly understand an affection involving the greater part if not the whole of the thickness of

the spinal cord. The patient has fever, tenderness and pain in the back, paralysis of the lower limbs, bladder, and rectum, gangrene and sloughing of the nates, loss of sensibility in the paralyzed limbs, with an exaggerated reflex action, and an unimpaired electric reaction in the same limbs. The present case is as remote as possible from such a group of symptoms.

4. Hæmorrhage in the cord, independently of myelitis, hardly occurs. Its consequences would be similar to those of acute myelitis. And it is very difficult to see how embolism could have produced the present group of symptoms.

5. The implication of the posterior columns of the cord, in any morbid change, should be followed by impairment of the power of coördinating muscular movements, or by loss of tactile sensibility, neither of which symptoms was present.

6. The posterior nerve-roots were certainly not affected.

7. The posterior gray matter of the cord. Lesion of this organ would involve a loss of the sense of pain.

8. The lateral white columns. Chronic sclerosis of these organs causes wasting of the muscles, and a loss of motor power; but this loss of power comes on quite slowly, and is usually accompanied by rigidity of certain muscles, and spontaneous pains, and formications, which did not occur in our case. The electric reaction, also, is retained till the very last in this complaint, the "progressive lateral sclerosis with muscular atrophy" of Charcot. In the presence of tremor and muscular wasting, and still more in the absence of fever, of complications of the bladder or rectum, and of eschars, the disease as described by Charcot resembles the present case; but his dictum is positive, that "wherever you meet with lateral sclerosis, contraction of the muscles is sure, sooner or later, to show itself in a more or less marked degree."[1] And contractions have not occurred in our case.

We have now narrowed our inquiry to the anterior cornua and columns of the spinal cord. Unless, indeed, we are inclined to seek in the peripheral nerves and the muscles for the causative lesion; but I think my audience will spare the argument upon this head, and suffer me to proceed directly to the statement of a distinct and nearly constant lesion, occurring in those parts of the spinal cord in which we have been led *a priori* to expect it. In nine of twelve[2] autopsies made upon children, the nerve-cells occupying the external portion of the anterior cornua, and the nervous reticulum formed by their prolongations, have been found atrophied; in the other three, the anterior roots were atrophied, and the anterior columns sclerosed. To this add, as more or less prom-

[1] London Lancet, August 1, 1874.

[2] For a *résumé* of literature, see Dr. Mary Putnam Jacobi's exhaustive article in the American Journal of Obstetrics for May, 1874.

inent accompaniments, proliferation of the connective nuclei, dilatation and fatty degeneration of blood-vessels, and distinct limited foci of softening, in the anterior cornua. In Gombault's case, occurring in an adult, granular degeneration of the ganglion cells of the anterior cornua was observed throughout the length of the cord, while the white substance and posterior gray substance were uninjured. For the sake of fairness it should be added that three apparently trustworthy examinations have been reported by Bouchut and Kétli, in which the spinal cord of children was found free from microscopical lesions. The evidence, however, largely preponderates in favor of that view of the pathology of the disease which permits us to name it an "inflammation of the motor tract of the spinal cord." [1]

It is needless to recapitulate the points of resemblance between "infantile paralysis" and the case I have related; in fact, the comparison has already been drawn, in the sketch of the disease with which the article opens. There are, however, some points of apparent disagreement. *Fever* often attends the onset in children; it was absent in this case, but among the similar cases hitherto reported in adults there are several in which fever was a very marked initial symptom. *Convulsions* sometimes occur in children; but they form so frequent an accompaniment of the acute diseases of infancy as to possess very little diagnostic importance. *Pain* in the back and limbs is another initial symptom, which was observed in a number of the adult cases. And the *deformity* which occurs so often in children is paralleled in several adult cases, as in No. LXXI. of Duchenne, in which the attack, brought on by exposure to cold, was followed by wasting of the flexors of one foot, with talipes equinus; as also in the one briefly mentioned by Dr. Jacobi,[2] with slight varus equinus and clawed hands.

Seguin has enumerated fifteen cases in adults, and has added six observed by himself. The latter exhibit symptoms of disturbance of sensation to a greater extent than is usual; and in one case the face, tongue, and muscles of the orbit were paralyzed, which has not been observed in other cases. The latest that has come to my knowledge is one by Frey, quite typical in every respect except its very gradual approach and progress; it is the one alluded to in the beginning of this article, under the name of "poliomyelitis." Of the list given by Seguin, I am compelled to reject Nos. VIII. and IX., cited from Moritz Meyer, whose account is excessively meagre; this leaves nineteen, or, with Frey's case and the one now given, twenty-one, of which twelve were acute and the others subacute or chronic.

In regard to the prognosis of this disease in adults, it is important that

[1] As proposed by Seguin in his recent able monograph, read before the New York Academy of Medicine, November 5, 1874, and printed in the Transactions of the Academy.

[2] Loco citato, pp. 21, 22.

the physician should clearly distinguish cases of ordinary myelitis, which it is not hard to do. What has been called "acute ascending paralysis" can with difficulty be distinguished at the outset from the present disease; its leading feature consists in a tendency to spread from one to another region of the spinal cord, either upwards or downwards, and to produce death in a week or two by paralysis of the muscles of respiration; if this tendency is not present, life will almost certainly be spared. Even in acute ascending cases, there is room to hope that the process may be arrested. As for the relief of local paralysis, the prospect is good in proportion as we succeed in making the muscles react under electrical stimulation.